

EDITORIAL COMMENT

Flow Propagation Analysis

Computer or Eyeball?*

James D. Thomas, MD

Cleveland, Ohio

Blood flow throughout the cardiovascular system is governed by the Navier-Stokes equations, a set of 4 coupled 4-dimensional (3 of space and 1 of time) partial differential equations. Although these are impossibly complex to solve analytically or even with the most powerful supercomputers, there are a number of places in the heart where simplification of these equations allows important clinical and research observations. The most commonly used of these is the simplified Bernoulli equation in echo-

[See page 37](#)

cardiography, where—for flow across a fixed stenosis—the relationship between pressure (P) in mm Hg, and velocity (v) in m/s, reduces to a single simple equation: $\Delta P = 4v^2$. A more complicated but related situation is flow across a normal mitral valve, where there is no abrupt pressure change but a gradual pressure gradient within the left ventricle (LV). Here again, echocardiography offers key data in the form of the color M-mode Doppler display, a spatiotemporal map of flow velocity along the echo scanline.

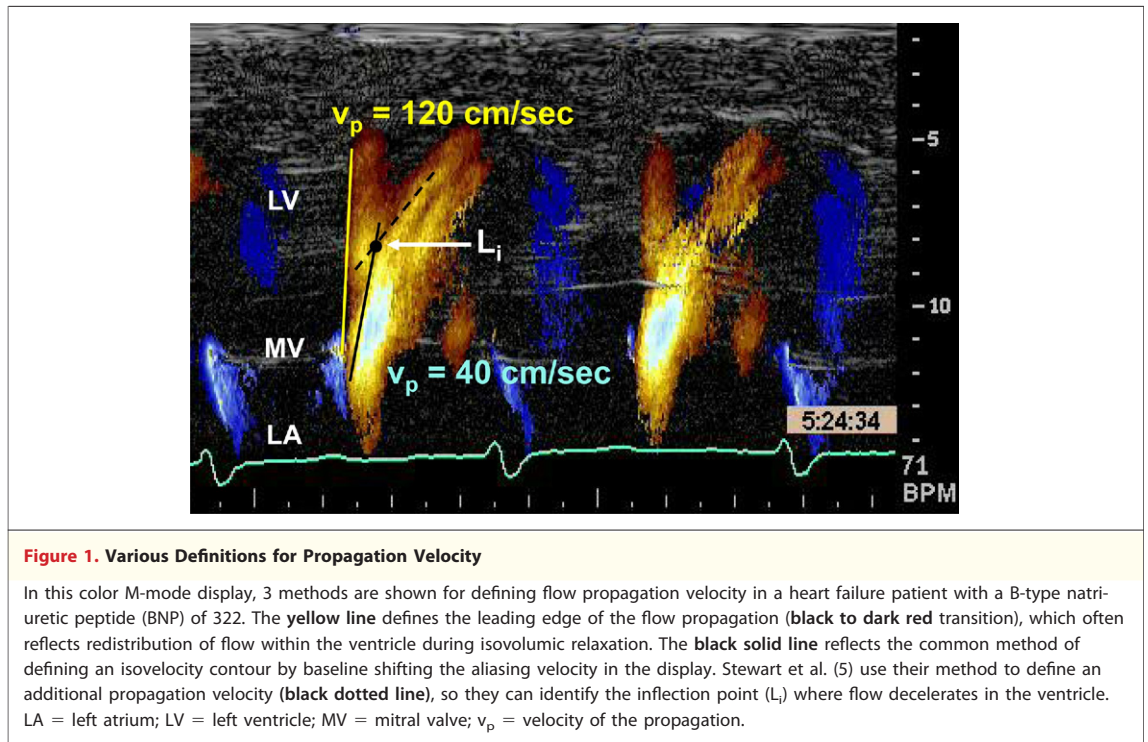
In 1992 Philippe Brun (1) described the use of color M-mode Doppler echocardiography to visualize flow propagation from the left atrium to the LV. In a series of manuscripts, Brun et al. (2) demonstrated that the velocity of the propagation (v_p) from atrium to ventricle was inversely related to the ventricular relaxation time constant: the more rapidly the ventricle relaxed, the more rapid the

flow propagation into the ventricle. In the intervening years, color M-mode flow propagation has achieved a place (although not a dominant one) (3) in the pantheon of parameters used to assess left ventricular diastolic dysfunction, but there has been controversy as to just how to define the flow propagation velocity. Initially (1), v_p was proposed to be the slope of the leading edge of the color Doppler (black to dark red transition), but this is often related to redistribution of blood within the ventricle during isovolumic relaxation and not reflective of actual blood propagation from the atrium to the ventricle (Fig. 1, yellow line). Later (4), it was suggested to highlight a single contour within the early part of flow propagation by setting the aliasing velocity at approximately 30% to 40% of the peak diastolic velocity of the E wave and measuring the slope of that contour (Fig. 1, black solid line). Normal patients typically have velocities >50 cm/s, whereas those with diastolic dysfunction have slower ones. Furthermore, the ratio of the peak E-wave velocity divided by this v_p provides a reasonable estimation of filling pressure (4).

Into this arena comes new work from Stewart et al. (5) in this issue of *JACC*, where they propose a more sophisticated analysis of the flow propagation data. In this approach, they identify not 1 but 2 distinct slopes of flow propagation. The first of these slopes is very close to what would currently be identified as v_p , whereas the second slope occurs near the end of the inflow profile (in spatial terms, usually near the middle of the ventricle), where the flow propagation abruptly slows as it begins to decelerate before reaching the apex of the heart (Fig. 1, black dotted line, which is only approximate, because the sophisticated software used in Stewart et al. [5] is not widely available). The key to their method is to identify the spatial location where this deceleration occurs, a point that they

*Editorials published in *JACC: Cardiovascular Imaging* reflect the views of the authors and do not necessarily represent the views of *JACC: Cardiovascular Imaging* or the American College of Cardiology.

From the Department of Cardiovascular Medicine, Heart and Vascular Institute, Cleveland Clinic, Cleveland, Ohio. The author has reported that he has no relationships to disclose.



show is closer to mitral annulus in patients with diastolic dysfunction than in those with normal diastolic function (L_i), identified in Figure 1. Overall, they demonstrate a modest slowing in v_p as well as a modest migration of the deceleration point L_i toward the mitral annulus as diastolic dysfunction worsens. However, because both of these relationships behave similarly, by multiplying the 2 together ($v_p \times L_i$), they achieve a new parameter which they term the strength of the propagation (v_s), which displays a stronger variation with grades of diastolic dysfunction than either of its components.

With good experimental technique, they initially developed this method in a group of 145 patients and then subsequently validated it in a separate group of 160 patients. In the validation cohort they used 3 different “gold” standards for identifying patients with diastolic dysfunction: 1) a pulmonary capillary wedge (PCW) pressure >18 (in 56 of the 160 patients); 2) a B-type natriuretic peptide (BNP) >100 pg/ml; and 3) a reduced intraventricular pressure difference (IVPD) <2.2 mm Hg. Interestingly, a side analysis of these data emphasizes just how frustrating the clinical assessment of diastolic function can be. When these 3 “gold” standards were compared against each other, they saw mutual classifications that were little better than a flip of

the coin: IVPD versus BNP = 57%; PCW versus BNP = 55%; and PCW versus IVPD = 65%!

The IVPD is a particularly interesting parameter, because it uses a more comprehensive analysis of the transmitral color M-mode flow data to calculate the actual pressure drop between the base and the apex of the LV during early diastolic filling, a phenomenon that can rightly be termed diastolic suction. By assuming that flow along the Doppler scanline is actually a streamline of flow, it is possible to estimate the pressure drop along this line by applying the Euler equation, which is essentially a 1-dimensional simplification of the Navier Stokes equations and can be thought of as a distributed version of the Bernoulli equation. Indeed, the measurement of the base to apex intraventricular pressure difference has been well-validated in a canine model (6), in patients undergoing septal myectomy (a particularly challenging geometry for this type of analysis) (7), and in patients with dilated cardiomyopathy (8). It has been applied to patients undergoing alcohol septal ablation for hypertrophic cardiomyopathy, demonstrating that the improvement in IVPD correlated with improved symptoms in these patients (7). The IVPD concept is not confined to diastole, because it can be applied to the left ventricular outflow tract during systole to estimate the ejection IVPD, which has been shown

to closely match invasive indexes of systolic function (9).

Returning to consideration of Stewart et al. (5), one might question the need for a new, numerically processed assessment of the color M-mode flow data. What will it add to the simple manual estimation of v_p or the more definitive calculation of IVPD via the Euler equation? First, one should recognize that IVPD and the new parameter v_s are quite different entities. Although Stewart et al. (5) have shown differences in v_s between grades of diastolic dysfunction in resting echocardiograms, IVPD shows its greatest difference during exercise. In a study of normal subjects and those with heart failure, there was no difference in IVPD at rest, but the ability to increase IVPD was the best predictor of maximal exercise tolerance (10). This finding was confirmed in patients with hypertrophic cardiomyopathy in a study that demonstrated the close connection between ventricular untwisting and the IVPD (11). Indeed the importance of augmented IVPD (diastolic suction) during inotropic stimulation of the heart was demonstrated nearly 20 years ago by Little (12), senior author of the current work (5). Thus, v_s and IVPD seem to give complementary information, and a method to estimate v_s would seem a worthy goal. A bigger issue, however, is whether the considerable computational challenges of measuring v_s are worth the effort in

comparison with simple manual measurement of v_p , which can be done in a matter of seconds with any echocardiography review software. Before this question can be answered, it will be necessary for the method of Stewart et al. (5) to be tested in other laboratories both for feasibility and utility. If its value can be shown in this setting, then what remains is for echo vendors to implement the algorithm within their review software. Although this would seem to be a reasonable expectation for any validated algorithm, echocardiography software developers have been surprisingly slow to implement automated analyses of color Doppler data for any purpose, whether it be to calculate the IVPD, to analyze the proximal convergence zone in valvular regurgitation, or even something as simple as calculating stroke volume from color flow in the left ventricular outflow tract. Although this writer hopes that vendors will implement the v_s algorithm to allow widespread testing, ideally this would be part of a comprehensive suite of validated color Doppler algorithms that exploit the physics of intracardiac flow to greatly expand the quantitative potential of echocardiography.

Reprint requests and correspondence: Dr. James D. Thomas, Cleveland Clinic, 9500 Euclid Avenue, Desk J1-5, Cleveland, Ohio 44195. *E-mail:* thomasj@ccf.org.

REFERENCES

1. Brun P, Tribouilloy C, Duval AM, et al. Left ventricular flow propagation during early filling is related to wall relaxation: a color M-mode Doppler analysis. *J Am Coll Cardiol* 1992;20:420–32.
2. Duval-Moulin AM, Dupouy P, Brun P, et al. Alteration of left ventricular diastolic function during coronary angioplasty-induced ischemia: a color M-mode Doppler study. *J Am Coll Cardiol* 1997;29:1246–55.
3. Nagueh SF, Appleton CP, Gillebert TC, et al. Recommendations for the evaluation of left ventricular diastolic function by echocardiography. *J Am Soc Echocardiogr* 2009;22:107–33.
4. Garcia MJ, Ares MA, Asher C, Rodriguez L, Vandervoort P, Thomas JD. An index of early left ventricular filling that combined with pulsed Doppler peak E velocity may estimate capillary wedge pressure. *J Am Coll Cardiol* 1997;29:448–54.
5. Stewart KC, Kumar R, Charonko JJ, Ohara T, Vlachos PP, Little WC. Evaluation of LV diastolic function from color M-mode echocardiography. *J Am Coll Cardiol Img* 2011;4:37–46.
6. Greenberg NL, Vandervoort PM, Thomas JD. Instantaneous diastolic transmitral pressure differences from color Doppler M mode echocardiography. *Am J Physiol* 1996;271:H1267–76.
7. Rovner A, Smith R, Greenberg NL, et al. Improvement in diastolic intraventricular pressure gradients in patients with HOCM after ethanol septal reduction. *Am J Physiol Heart Circ Physiol* 2003;285:H2492–9.
8. Yotti R, Bermejo J, Antoranz JC, et al. A noninvasive method for assessing impaired diastolic suction in patients with dilated cardiomyopathy. *Circulation* 2005;112:2921–9.
9. Yotti R, Bermejo J, Desco MM, et al. Doppler-derived ejection intraventricular pressure gradients provide a reliable assessment of left ventricular systolic chamber function. *Circulation* 2005;112:1771–9.
10. Rovner A, Greenberg NL, Thomas JD, Garcia MJ. Relationship of diastolic intraventricular pressure gradients and aerobic capacity in patients with diastolic heart failure. *Am J Physiol Heart Circ Physiol* 2005;289:H2081–8.
11. Notomi Y, Martin-Miklovic MG, Oryszak SJ, et al. Enhanced ventricular untwisting during exercise: a mechanistic manifestation of elastic recoil described by Doppler tissue imaging. *Circulation* 2006;113:2524–33.
12. Cheng CP, Igarashi Y, Little WC. Mechanism of augmented rate of left ventricular filling during exercise. *Circ Res* 1992;70:9–19.

Key Words: color M-mode echocardiography ■ flow propagation ■ left ventricular diastolic function.